

## Carotid sinus syncope

A LEATHAM

*From St. George's and The National Heart Hospitals, London*

Every physician knows about Soma Weiss's Boston tram-driver who repeatedly blacked out on a corner from stimulation of the carotid sinus by his wing collar.<sup>1</sup> Further cases of so-called carotid sinus syncope have only rarely been reported. It is therefore surprising to see that Morley and his colleagues<sup>2</sup> describe 70 patients with carotid sinus syncope who have been treated by artificial pacing over a period of four years. This raises the problem of the definition of carotid sinus syncope. Reflex slowing of sinoatrial rate from carotid sinus massage is an almost universal finding and is perhaps more pronounced in older subjects and certainly is particularly noticeable, as one would expect, in patients with disease of the conducting tissue. Presumably Morley *et al.* are raising the interesting idea that the slowing in their cases is the result of a primary abnormality of the carotid sinus, but others would localise the primary abnormality to the conducting tissue, and in patients with sinus bradycardia and sinus pauses (but no evidence of atrioventricular conduction problems) would regard the primary diagnosis as that of sinoatrial disease. This is now known to be a very frequent abnormality and is indeed sometimes hard to differentiate from normal subjects with strong vagal tone unless they have severe symptoms or attacks of atrial fibrillation or flutter.

How then do Morley *et al.* differentiate sinoatrial disease from so-called carotid sinus syncope? In the first place they recognise that over half their patients had positive evidence of sinoatrial disease based on the presence of paroxysmal atrial fibrillation and a prolonged sinoatrial node recovery time, though the latter is of doubtful validity.<sup>3</sup> But they go on to state that "none of the patients had overt sick sinus syndrome on resting or 24 hour electrocardiogram". This seems a remarkable statement when one commonly sees patients with episodes of extreme bradycardia separated by weeks or months of normality.

Thus a strong case can be made for stating that most, if not all, of the patients described by Morley *et al.* simply had sinoatrial disease. The question then

arises, knowing the benign course of this disease unlike atrioventricular conduction disorders, whether too many cases of this sort are being paced, and one strongly suspects that this is so particularly in the United States where 40% of all cases paced are paced primarily for sinoatrial disease, according to the World Survey of Cardiac Pacing in 1978.

In this country patients and physicians are more stoical about minor dizzy turns, particularly when months, or even years, go by without recurrence. It is, however, important to avoid drugs since some of these patients also have tachyarrhythmias (brady-tachy syndrome) which will be exacerbated by adrenergic therapy (they are also liable to thromboembolism and may require anticoagulants<sup>4</sup>). In the few whose symptomatology is sufficiently severe to require pacing, suppressant drugs can then be given safely. In "early cases" on a pacemaker when the patient is in sinus rhythm with a reasonable rate most of the time, the occasional onset of bradycardia stimulating demand ventricular pacing is often noticed by the patient because of a slight fall in blood pressure, with loss of appropriate presystolic atrial contraction, and so it is not surprising that some of Morley's cases continue to have symptoms from the so-called "pacemaker effect". Whether Morley *et al.* are correct in thinking that their patients have an additional problem described as a vasodepressor response indicating an abnormal fall of blood pressure after carotid sinus massage when they only had 11 control subjects remains to be decided. Atrial pacing was used in eight of their cases, but had to be discontinued, in some because of coincidental atrioventricular block and in others because of atrial arrhythmia, both problems being expected in patients with sinoatrial disease. It is interesting to read that of the 16 patients with persistent symptoms despite advanced pacing systems, 12 had adequate bradycardia control. It is unlikely that they were suffering from vasomotor syncope from carotid sinus pressure from winged collars, and maybe their symptoms had another cause.

Further work is required to decide whether prim-

ary carotid sinus syncope is other than an extremely rare medical curiosity. In any case the estimate of 35 new patients per million per year of "carotid sinus syndrome" in the Worthing area compared with 82 new patients with atrioventricular block and sick sinus syndrome must be an exaggeration. In the meantime carotid sinus stimulation may be regarded as a means of unmasking conduction disease, though the subject is made difficult by the enormous range of physiological variation. Serious thought should also be given to whether pacing is being too frequently used for patients with sinoatrial disease. Furthermore it seems that there is seldom justification for the use of expensive sequential atrioventricular systems, particularly when most of these patients have unstable electrical activity in the atria, though modern techniques may, at least in part, overcome this problem in the future.<sup>5</sup>

#### References

- 1 Weiss S, Capps RB, Ferris EB, Jr, Munro D. Syncope and convulsions due to a hyperactive carotid sinus reflex: diagnosis and treatment. *Arch Intern Med* 1936; 58: 407-17.
- 2 Morley CA, Perrins EJ, Grant P, Chan SL, McBrien DJ, Sutton R. Carotid sinus syncope treated by pacing. Analysis of persistent symptoms and role of atrioventricular sequential pacing. *Br Heart J* 1982; 47:
- 3 Evans TR, Callowhill EA, Krikler DM. Clinical value of tests of sino-atrial function. *PACE* 1978; 1: 2-7.
- 4 Fairfax AJ, Lambert CD, Leatham A. Systemic embolism in chronic sino-atrial disorder. *N Engl J Med* 1976; 295: 190-2.
- 5 Barold SS, Falkoff MD, Ong LS, Heinle RA. The third decade of cardiac pacing. Multiprogrammable pulse generators. *Br Heart J* 1981; 45: 357-64.

Requests for reprints to Dr Aubrey Leatham, National Heart Hospital, Westmoreland Street, London W1M 8BA.